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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/683,815	10/09/2003	Arnon Rosenthal	514712001500	5142

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EXAMINER	
BALLARD, KIMBERLY A	
ART UNIT	PAPER NUMBER
1649	

SHORTENED STATUTORY PERIOD OF RESPONSE	MAIL DATE	DELIVERY MODE
3 MONTHS	02/07/2007	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

If NO period for reply is specified above, the maximum statutory period will apply and will expire 6 MONTHS from the mailing date of this communication.

Office Action Summary	Application No. 10/683,815	Applicant(s) ROSENTHAL ET AL.	
	Examiner Kimberly A. Ballard	Art Unit 1649	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *20 December 2006*.
2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.
3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1,2,4-14,16-27,29-38 and 51-53* is/are pending in the application.
4a) Of the above claim(s) _____ is/are withdrawn from consideration.
5)☐ Claim(s) _____ is/are allowed.
6)☒ Claim(s) *1,2,4-14,16-27,29-38 and 51-53* is/are rejected.
7)☐ Claim(s) _____ is/are objected to.
8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.
10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
a)☐ All b)☐ Some * c)☐ None of:
1.☐ Certified copies of the priority documents have been received.
2.☐ Certified copies of the priority documents have been received in Application No. _____.
3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).
* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08)
Paper No(s)/Mail Date *12/20/06, 01/24/07*.
4) ☐ Interview Summary (PTO-413)
Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Status of Application, Amendments and/or Claims

1. A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submission filed on December 20, 2006 has been entered.

2. In the amendment filed December 20, 2006, Applicant has amended claims 1, 13 and 26 and has canceled claims 3, 15, 28, 39-50 and 54. Following the amendment, claims **1, 2, 4-14, 16-27, 29-38,** and **51-53** are pending and under examination in the instant office action.

3. Any objection or rejection of record of claims 3, 15, 28, 39-50 and 54 is hereby rendered moot in view of Applicant's cancellation of said claims.

4. The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

Information Disclosure Statement

5. Signed and initialed copies of the IDS papers submitted December 20, 2006 and January 24, 2007 are enclosed in this action. The Examiner has included copies of IDS papers from September 27, 2005 and May 1, 2006, wherein certain references that had been inadvertently and regretfully not previously considered have now been considered.

Withdrawn Objections and Claim Rejections

6. The rejection of claims 1, 2, 4, 6, 8-10, 13, 14, 16, 18, 20-22, 25-27, 29, 31, 33-35, 38, 42, 44, and 46-48 under 35 U.S.C. 102(b) as being unpatentable over Bard et al. (*Nat Med*, August 2000; 6(8): 916-919) as set forth at ¶ 10 of the previous office action (07/21/2006) is withdrawn in view of Applicant's amendments to the claims.

7. The rejection of claims 1, 2, 8-14, 20-27, 33-38, and 46-53 under 35 U.S.C. 102(e) as being anticipated by US Patent No. 6,787,523 B1 to Schenk as set forth at ¶ 11 of the previous office action (07/21/2006) is withdrawn in view of Applicant's amendments to the claims.

8. The rejection of claims 1, 2, 4-14, 16-27, 29-38 and 51-53 under 35 U.S.C. 103(a) as being unpatentable over Bard et al. (*Nat Med*, August 2000, 6(8): 916-919) in view of Zeitlin et al. (*Microbes and Infection*, 2000, 2: 701-708) as set forth at ¶ 12 of the previous office action (07/21/2006) is withdrawn in view of Applicant's amendments to the claims.

Maintained Claim Rejections

Obviousness-Type Double Patenting

9. The provisional rejection of instant claims 1, 2, 4, 6, 10-14, 16, 18, 22-24, 26, 27, 29, 31, 35-37 and 51-53 over claims 1, 2, 4, 7-17, 19, 28, 30-40 and 42 of copending Application No. 11/194,989 is maintained for reasons of record set forth in the office action dated 01/31/2006 and held in abeyance until all other rejections are resolved.

10. The provisional rejection of instant claims 1, 2, 4, 6, 10-14, 16, 18, 22-24, 26, 27, 29, 31, 35-37 and 51-53 over claims 1-3, 6-15, 17, 26, 28-38 and 40 of copending Application No. 11/195,207 is maintained for reasons of record set forth in the office action dated 01/31/2006 and held in abeyance until all other rejections are resolved.

New Claim Rejections and/or Objections

Claim Objections

11. Claims 8, 9, 20, 21, 33 and 34 are objected to because of the following informalities: the phrase "a monoclonal antibody comprising amino acid sequence of SEQ ID NO: 4 and 6" appears to contain typographical errors, resulting in the phrase sounding grammatically awkward. The Examiner kindly suggests the following substitute phrase: "a monoclonal antibody comprising the amino acid sequences of SEQ ID NOs: 4 and 6". Appropriate correction is required.

Obviousness-Type Double Patenting

12. Claims 1, 2, 10, 13, 14, 22, 26, 27 and 35 are provisionally rejected on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims 41-43 of copending Application No. 11/413,817 (hereinafter the '817 application). Although the conflicting claims are not identical, they are not patentably distinct from each other because the '817 application contains claims drawn to methods of: treating Alzheimer's disease in a subject, suppressing formation of amyloid plaques in a subject, and reducing amyloid plaques in a subject, each method comprising the administration of an effective amount of a pharmaceutical comprising a monoclonal antibody that specifically binds to an epitope on $A\beta_{1-40}$ that includes amino acids 25-34 and 40, wherein the antibody binds to $A\beta_{1-40}$ with higher affinity than $A\beta_{1-42}$ and $A\beta_{1-43}$, and wherein the antibody is not antibody 2294. Accordingly, the claims of the '817 application render obvious instant claims 1, 2, 10, 13, 14, 22, 26, 27 and 35.

This is a provisional obviousness-type double patenting rejection because the conflicting claims have not in fact been patented.

Claim Rejections - 35 USC § 112, first paragraph

13. The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

14. Claims 1-12 and 51 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method of administering an effective amount of a pharmaceutical composition comprising an antibody that binds specifically to amino acids 28-40 of $A\beta_{1-40}$ peptide (SEQ ID NO: 1) and a pharmaceutically acceptable carrier to a subject in need of reduction of brain amyloid plaques, wherein the antibody does not show significant cross-reactivity with $A\beta_{1-42}$ and $A\beta_{1-43}$ peptide, and wherein said administration results in reduction of amyloid plaques in the brain of said subject, does not reasonably provide enablement for a method of treating Alzheimer's disease as broadly claimed. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to practice the invention commensurate in scope with these claims.

The factors to be considered in determining whether a disclosure would require undue experimentation include (1) the quantity of experimentation necessary, (2) the amount of direction or guidance presented, (3) the presence or absence of working examples, (4) the nature of the invention, (5) the state of the prior art, (6) the relative skill of those in the art, (7) the predictability or unpredictability of the art and, (8) the breadth of the claims. *In re Wands*, 8 USPQ2d, 1400 (CAFC 1988).

The claims are drawn to a method for treating Alzheimer's disease in a subject. The instant specification defines “treatment” or “treating” as an approach for obtaining beneficial or desired clinical results including, but not limited to: inhibiting or suppressing the formation of amyloid plaques, reducing, removing, or clearing amyloid plaques, improving cognition or reversing cognitive decline, sequestering soluble Aβ peptide circulating in biological fluids, reducing Aβ peptide (including soluble and deposited) in a tissue (such as brain), inhibiting and/or reducing accumulation of Aβ peptide in the brain, inhibiting and/or reducing toxic effects of Aβ peptide in a tissue (such as brain), decreasing symptoms resulting from the disease, increasing the quality of life of those suffering from the disease, decreasing the dose of other medications required to treat the disease, delaying the progression of the disease, and/or prolonging survival of patients (see [0064]). Accordingly, the claimed treatment of Alzheimer's disease broadly encompasses such clinical results as “reversing cognitive decline” and “decreasing symptoms resulting from the disease”, results which are not fully represented by the instant specification.

The nature of the invention is the demonstration that administration of an anti-Aβ monoclonal 2286 antibody (directed to amino acids 28-40 of Aβ peptide) results in a decrease in brain amyloid-β (Aβ) pathology (i.e., reduced amyloid deposits and thioflavine-S positive compact plaques) and increased MHC-II staining (indicating microglial activation) in transgenic Tg2576 mice, which are an animal model of Alzheimer's disease (Example 5). The brains of the Tg2576 mice injected peripherally or intracranially with monoclonal anti-Aβ antibodies were evaluated histopathologically 3 days following treatment. Applicant also demonstrates that chronic administration of monoclonal antibody 2286 to Tg2576 transgenic mice elevates both Aβ serum concentrations as well as titers of anti-Aβ antibodies in the serum, suggesting that the antibody may have changed Aβ equilibrium between CNS and plasma, possibly facilitating clearance of Aβ out of the CNS (Example 7). However, no examples, prophetic or otherwise, of the effects of anti-Aβ antibodies on the treatment of other clinical symptoms associated with Alzheimer's disease other than amyloid-associated pathology in the brain or circulation are provided.

The state of the art recognizes that Alzheimer's disease has many features, including short-term memory loss, behavioral abnormalities, as well as a multitude of anatomical abnormalities such as the presence of both amyloid plaques and neurofibrillary tangles comprising the tau protein. See for example Small et al. *Proc Natl Acad Sci USA*, 2000; 97:6037-6042. Additionally, Alzheimer's disease is characterized by changes in permeability of the blood brain barrier; see Anderson (U.S. Patent 5,589,154), particularly column 6 lines 27 - 40, where the reference teaches that beta-amyloid protein, the causative agent in Alzheimer's diseases, also induces vascular damage. Applicant fails to provide guidance on the treatment of these other features associated with Alzheimer's disease pathology, which would be encompassed by treatment of AD. Moreover, both at the time of filing and now, effective therapy for the treatment and prevention of Alzheimer's has eluded researchers. De Lustig et al. (*Rev in Neurosciences*, 1994; 5: 213-225, cited previously) report that there is still no adequate preventive strategy and no effective therapies for the pathology, and the disease thus follows an inevitable degenerative course. And a recent review by Vickers (*Drugs Aging*, 2002, 19(7): 487-494, cited previously) notes that there is no effective treatment currently available to reverse, slow down or prevent the course of Alzheimer's disease and most other brain diseases and neurodegenerative conditions.

The instant specification hence teaches reduction of amyloid brain pathology in transgenic mice, but insufficient guidance is provided for treatment of Alzheimer's disease as broadly claimed. Moreover, the animal model used in the instant specification is not recognized as providing for teachings that are predictive of the results that would be expected for the full scope of the claims. For example, the art recognizes that such *in vivo* transgenic animal models are not readily correlated to the human *in vivo* case. In particular, the art teaches a lack of correlation of beneficial effects shown in the mouse model system in humans; see in particular Münch & Robinson (*J. Neural Transmission,* July 2002; 109(7-8): 1081-1087, cited previously). And while the art may recognize that passive immunization with anti-Aβ antibodies leads to beneficial results in animal models of Alzheimer's disease (AD) (see Bard et al. *Nature Med.* 2000; 6(8):916-919, listed on Applicant's IDS), the art also acknowledges that many issues still need to be resolved in immunotherapy of AD in humans (see, for example, De Felice & Ferreira, *Cell Mol Neurobiol.* 2002; 22(5-6): 545-563; and St. George-Hyslop et al. *Nature*, 1999; 400:116-117). For example, although immunization with Aβ peptide and passive immunization with anti-Aβ antibodies have been shown to be effective in transgenic murine models of AD, clinical trials using Aβ peptide vaccines in AD patients have revealed serious negative side effects of the immunotherapy in a number of the patients, such as inflammation of the CNS, which is indicative of a potential breakdown of the blood-brain barrier and entry of T-cells into the brain (see p. 551 of De Felice & Ferreira 2002; and Münch & Robinson 2002). The art to which the present invention relates is therefore highly unpredictable and unreliable with respect to conclusions drawn from laboratory data extrapolated to clinical efficacy.

Due to the large quantity of experimentation necessary to determine the effectiveness of treatment of Alzheimer's disease symptoms or of reversing cognitive decline in the human subjects, the lack of direction/guidance presented in the specification regarding the same, the complex nature of the invention, the state of the prior art with respect to treatment of Alzheimer's, the unpredictability of immunotherapy as evidenced in the art, and the breath of the claims which effectively fail to limit a specific clinical symptom, undue experimentation would be required of the skilled artisan to make and/or use the claimed invention in its full scope.

Claim Rejections - 35 USC § 112, second paragraph

15. The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

16. Claims 1, 2, 4-14, 16-27, 29-38 and 51-53 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

The term "binds preferentially" in claims 1, 2, 13, 14, 26 and 27 is a relative term which renders the claim indefinite. The term "preferentially" is not defined by the claim, the specification does not provide a standard for ascertaining the requisite degree, and one of ordinary skill in the art would not be reasonably apprised of the scope of the invention. The art accepted term for antibody binding is “specific binding”. It is noted that incorporation of a phrase such as "binds specifically" or "specifically binds" into the claims would negate this rejection.

Claim Rejections – 35 USC § 102

17. Claims 1, 2, 4-14, 16-27, 29-38 and 51-53 are rejected under 35 U.S.C. 102(e) as being anticipated by US Patent Application No. 2003/0073655 A1 by Chain, published April 17, 2003, filed February 28, 2002 (on Applicant's IDS), as evidenced by Zeitlin et al. (*Microbes Infection*, 2000; 2: 701-708; cited in previous office action).

Chain teaches methods of treating a subject having Alzheimer's disease or another disease or disorder characterized by amyloid β deposition, comprising administering a pharmaceutical composition comprising an antibody targeted to β amyloid peptide, wherein the antibody is free-end specific for the C-terminus of Aβ peptide (see [0009]). Similarly, Chain teaches methods for suppressing the accumulation of Aβ peptide comprising administering said antibody (see [0012]). Chain notes that Alzheimer's disease (AD), which is a uniquely human disease, is characterized by the presence of amyloid deposits within the brain (see [0003 and 0121]). Thus, Chain teaches that the principal goal of drugs designed to the treatment of AD is to reduce the amount of Aβ deposits in the central nervous system (CNS), which entails suppressing the production of Aβ, aiding in the clearance of Aβ, and/or inhibiting the formation of insoluble Aβ fibrils (see [0005]). Accordingly, Chain's disclosed methods of treating AD and suppressing Aβ accumulation would encompass a method of reducing amyloid plaques in the brain of a subject, particularly a human subject, and would thus address recited limitations within instant claims 1, 13, 25, 26, 38 and 51-53.

Chain discloses that the most effective target for end-specific antibodies as therapy for AD is likely to be Aβ1-40, which forms the bulk of circulating Aβ peptide in human CSF, plasma and urine (see [0041]). As such, the free-end specific C-terminal antibodies are disclosed by Chain are targeted to Aβ1-40, which antibody does not bind to the amyloid β-precursor protein (APP) from which it is derived (see [0071]), meaning that such antibodies will bind to a peptide ending at residue 40 of Aβ, but will not bind to a peptide having residues 41, 42, or 43 of Aβ (see also [0064]). For example, Chain discloses that the free end specific antibody is targeted to amino acids 33-40 (or 34-40 or 36-40 or 37-40) of the C-terminus-truncated Aβ-peptide in addition to a free carboxylic (COOH) group (see [0076]). Such an antibody epitope includes amino acids 39 and/or 40 of $A\beta_{1-40}$, thus addressing limitations regarding the antibody binding recited limitations of instant claims 2, 14 and 27, and would similarly anticipate the preferential binding to amino acids 28-40 of $A\beta_{1-40}$ recited in instant claims 1, 13 and 26.

Chain discloses that the administered antibody is a monoclonal antibody, a humanized antibody, a chimeric antibody, a bispecific antibody, a single chain (scFv) antibody, or a Fab fragment (see [0033] and claims), thus meeting recited limitations of instant claims 10, 11, 22, 23, 35 and 36. Chain also discloses that for the production of antibodies, human hosts may be utilized, which would generate human antibodies (see [0082]). Thus Chain teaches human antibodies as in instant claims 12, 24 and 37. Although Chain is silent with respect to the affinity of the monoclonal antibodies for $A\beta_{1-}$

$_{40}$, monoclonal antibodies are recognized in the art as possessing high specificity and binding affinity, typically in the range of 10^{-8} to 10^{-10} M, as evidenced by Zeitlin et al. (see pp. 701-702). Since 1 nM = 10^{-9} M, the monoclonal antibodies taught by Chain (and their Fab binding fragments) would inherently possess affinities for $A\beta_{1\text{-}40}$ of about 1 nM or less, thus addressing limitations recited in instant claims 4-7, 16-19 and 29-32.

As the recited binding limitations have been met, it would be an expected property of the disclosed monoclonal anti-$A\beta_{1\text{-}40}$ antibodies to competitively inhibit the binding of other monoclonal antibodies, such as the monoclonal antibody of instant claims 8, 20 and 33 (mAb 2286), which the instant specification discloses binds to an epitope which includes at least residues 39 and/or 40 of the $A\beta_{1\text{-}40}$ peptide and comprises at least residues 36-40 of $A\beta_{1\text{-}40}$ (see [0184] and [0187] of the instant application). Accordingly, the limitations of instant claims 9, 21 and 34, reciting that the antibody binds to the same epitope to which a monoclonal antibody comprising the amino acid sequences of SEQ ID NOs: 4 and 6 binds, would similarly be met. As such, the teachings of Chain fully anticipate instant claims 1, 2, 4-14, 16-27, 29-38 and 51-53.

18. Claims 1, 2, 4-14, 16-27, 29-38 and 51-53 are rejected under 35 U.S.C. 102(e) as being anticipated by US Patent Application No. 2004/0241164 A1 by Bales et al., published December 2, 2004, filed August 14, 2002 (on Applicant's IDS).

Bales et al. disclose treatment of conditions and diseases related to the amyloid-β (Aβ) peptide, such as Alzheimer's disease, Down's syndrome, cerebral amyloid angiopathy, mild cognitive impairment and the like (see [0002]). The method of treatment comprises administering to the subject an antibody that recognizes Aβ, wherein the antibody has greater affinity for soluble Aβ than 10^{-9} M, and more particularly, the affinity is with respect to $A\beta_{1-40}$ or $A\beta_{1-42}$ (see [0008]). Bales also discloses that the invention includes a method for reducing disease progression in a subject having a condition or disease associated with Aβ, comprising administration of an effective amount of an anti-Aβ antibody that has greater affinity for soluble $A\beta_{1-40}$ or $A\beta_{1-42}$ than 10^{-9} M (see [0009]). More particularly, affinities higher than 10^{-10} M or 10^{-11} M are preferred (see [0033]), which disclosed affinities are noted to be less than the instantly claimed 200 nM and 1 nM. Bales notes that the expression "Aβ40" is used to denote peptides that bind to antibodies that bind only at an Aβ C-terminus that ends at position 40 (see [0033]). For example, Bales discloses administration of the monoclonal antibody 2G3, which recognizes Aβ40 but not Aβ42, to transgenic PDAPP mice (these mice are an art-accepted animal model of Alzheimer's disease) (see [0174]). Because of the exquisite specificity in recognizing the 1-40 form of Aβ over the 1-42 form, logic would dictate that the epitope for these antibodies would reside in the C-terminus of Aβ, and would comprise residues 28-40 of Aβ, and more particularly, would include amino acids 39 and/or 40 of the $A\beta_{1-40}$ peptide. Accordingly, it would be an expected property of the disclosed high affinity monoclonal anti-$A\beta_{1-40}$ antibodies to competitively inhibit the binding of other monoclonal antibodies, such as the monoclonal antibody of instant claims 8, 20 and 33 (mAb 2286), which the instant specification discloses binds to an epitope which includes at least residues 39 and/or 40 of the $A\beta_{1-40}$ peptide and comprises at least residues 36-40 of Aβ_{1-40} (see [0184] and [0187] of the instant application). The antibodies disclosed by Bales would thus meet the recited antibody limitations of instant claims 1, 2, 4-7, 13, 14, 16-19, 26, 27 and 29-32. Additionally, the limitations of instant claims 9, 21 and 34, reciting that the antibody binds to the same epitope to which a monoclonal antibody comprising the amino acid sequences of SEQ ID NOs: 4 and 6 binds, would similarly be met. Bales also discloses pharmaceutical preparation of the antibodies, including pharmaceutical compositions comprising the antibodies and pharmaceutically acceptable excipients (i.e., carriers) (see [0157]), thus addressing another recited limitation within instant claims 1, 13 and 26.

Bales et al. teach that by "condition or disease related to Aβ" is meant conditions or diseases that are associated with: 1) the development of β-amyloid plaques in the brain, 2) the synthesis of abnormal forms of Aβ, 3) the formation of particularly toxic forms of Aβ, or 4) abnormal rates of synthesis, degradation, or clearance of Aβ (see [0027]). Use of the disclosed pharmaceutical compositions comprising anti-Aβ antibodies in the disclosed methods of treating Alzheimer's disease and reducing disease progression would therefore necessarily encompass and anticipate the instantly claimed methods of treatment of Alzheimer's disease, suppression of formation of amyloid plaques, and reduction of amyloid plaques in a subject of instant claims 1, 13, 25, 26 and 38. Bales also discloses that by "subject" is meant a mammal, preferably a human (see [0022]), thus anticipating instant claims 51-53.

Finally, the antibodies taught by Bales et al. include monoclonal antibodies, humanized antibodies and human antibodies (see [0038]), thus addressing recited limitations of dependent claims 10-12, 22-24 and 35-37. Accordingly, the teachings of Bales et al. fully anticipate instant claims 1, 2, 4-14, 16-27, 29-38 and 51-53.

Conclusion

19. No claims are allowed.

Advisory Information

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Kimberly A. Ballard whose telephone number is 571-272-4479. The examiner can normally be reached on Monday-Friday 9AM - 5:30PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Janet Andres can be reached on 571-272-0867. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

Elizabeth C. Kemmerer

Kimberly Ballard, Ph.D.
January 29, 2007

ELIZABETH KEMMERER
PRIMARY EXAMINER